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Research Interests

In inflammation, the recruitment of inflammatory cells from the blood into virtually every tissue is regulated by the interaction of adhesion molecules and chemokines/receptors expressed on the cell surface or released by leukocytes and endothelial cells. Crohn's disease (CD), a chronic inflammatory bowel disease, affects more than 1 million people in North America and poses an economic burden in excess of $2 billion per year. Granulocytes, monocytes, and lymphocytes are active participants in the chronic inflammatory process; however, granulocytes and monocytes migrate and localize to inflamed tissues, whereas lymphocytes acquire memory and recirculate thousands of times, back to areas with a similar microenvironment to that where they first recognized their cognate antigen. It is, therefore, likely that a subset of these cells is ultimately responsible for the relapse and perpetuation of disease in patients who have experienced clinical remission. Our laboratory is interested in understanding the redundant adhesive pathways that explain how these cells continue to migrate to the gut after the blockade or deletion of the crucial gut-homing adhesion molecule (i.e., integrin a4b7). We work with two unique murine models of CD that, similar to the human condition, develop predominant, chronic small intestinal inflammation. Our ultimate goal is to dissect the adhesive pathways that regulate lymphocyte migration in the chronically inflamed small intestine. Given the similarities between the chronic intestinal inflammation in our mouse models and that present in human CD, our studies may provide important leads for new biological targets to treat this devastating disease.

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